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Case Report

A case of an airway obstruction secondary to blood clot formation after an episode of massive hemoptysis in a smear positive pulmonary tuberculosis pregnant lady

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ABSTRACT

Here we report an interesting case of airway obstruction secondary to blood clot formation after an episode of massive hemoptysis in a pregnant lady and removal of clot with rigid bronchoscopy after an attempt of failure with flexible bronchoscopy with review of literature.

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1. Introduction

Pulmonary tuberculosis is a common cause for hemoptysis. Up to one third of patients with pulmonary tuberculosis develops hemoptysis during their illness.⁶ Before the advent of antibiotic therapy, 4–7% of tuberculosis related deaths were due to massive hemoptysis.⁷ In approximately 80% of the cases, the hemorrhagic source is found in the bronchial or other systemic arteries, supplying the lungs and in the remaining 20%, in the pulmonary arteries.⁸ Airway obstruction secondary to blood clot formation after massive hemoptysis is also reported.¹ Here, I am presenting an interesting case of a pregnant lady, suffering from pulmonary tuberculosis, who was on anti tubercular treatment for 2 weeks, had a complete collapse of left lung after an episode of hemoptysis, at the time of delivery. Blood clot causing complete collapse of the left lung was successfully removed by the rigid bronchoscopy after an attempt of failure by the flexible bronchoscopy. Mechanism of hemoptysis in tuberculosis, any possible effect of drugs (ethamsylate) and hypercoagulable state of pregnancy and tuberculosis^{2,3,5,12} in the rapid formation of blood clot and methods of its removal are discussed in this paper with the review of literature.

2. Case report

A 20-year-old pregnant lady was referred to chest clinic for the management of the hemoptysis. She was 36-weeks pregnant and

her medical history was negative for tuberculosis. She had symptoms of cough with mild hemoptysis, fever, and loss of appetite for 20 days. Her physical examination revealed fever and tachycardia. On auscultation, bilateral vesicular breath sounds were present with crackles present over the left mammary area. Chest radiograph (Fig. 1) was taken with abdominal guard, which showed infiltrations in the left upper zone, suggestive of pulmonary tuberculosis (PTB). Her blood mixed sputum came positive for Acid Fast Bacilli (AFB). Her Clotting Time was 2 min 45 s (normal 5–15 min), Activated Partial Thromboplastin Time (APTT) was 58 s (control 54 s), International Normalised Ratio (INR) was 1.2 (normal 0.9–1.2) and platelets were $200 \times 10^9/L$ (normal $150\text{--}400 \times 10^9/L$). No signs of superficial or deep vein thrombosis were present. She was started on anti tubercular treatment (ATT) with symptomatic and supportive treatment. After one week of therapy, further hemoptysis did not recur. She delivered a healthy male child after two weeks but had one episode of massive hemoptysis (>200 mL blood) during the delivery. Injection ethamsylate 250 mg IV 6 hourly with moist oxygen inhalation (2–3 L/min) was administered during this period. Chest radiograph (Fig. 2) after delivery showed complete collapse of the left lung. The flexible bronchoscopy was performed under 4% local xylocaine spray. Complete obstruction of the left main bronchus was observed due to the presence of a large blood clot, which could not be removed with the flexible bronchoscopy. The rigid bronchoscopy under short general anaesthesia was planned and the blood clot was successfully removed without any complication. Repeat chest radiograph (Fig. 3) done later, showed partial expansion of the collapsed lung. The patient was discharged after two days, with no new episode of hemoptysis.

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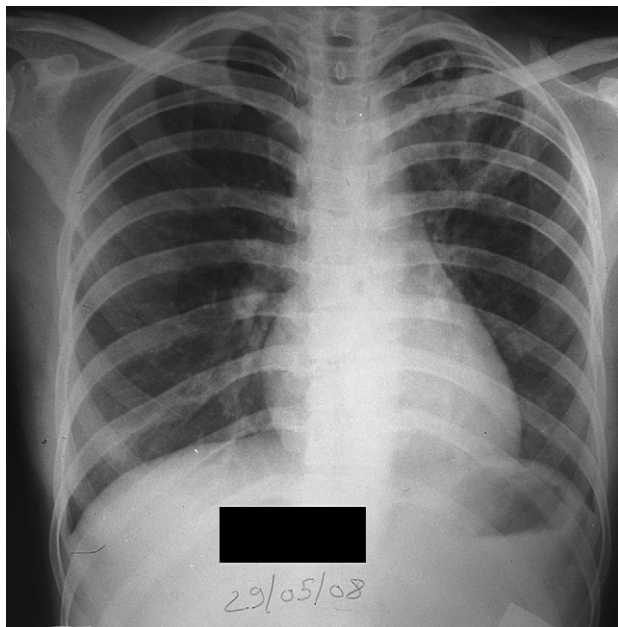


Fig. 1. Left upper zone infiltrations suggestive of pulmonary tuberculosis.

Follow up radiograph after one month, showed complete recovery with marked resolution of infiltrates and complete expansion of lung on the left side (Fig. 4).

3. Discussion

Inflammatory response and destruction due to the spread of tuberculosis into the adventitia and media of pulmonary or bronchial artery walls, results in the weakening of the arterial wall, allowing the development of a herniation of the vessel into the lumen of the cavity (pseudoaneurysm).⁹ The risk of rupture is then high due to the development of the aneurysm^{6–8} and the inflammatory processes in the arterial wall. The presence of a definite aneurysm has been the exception instead of the rule in the routine

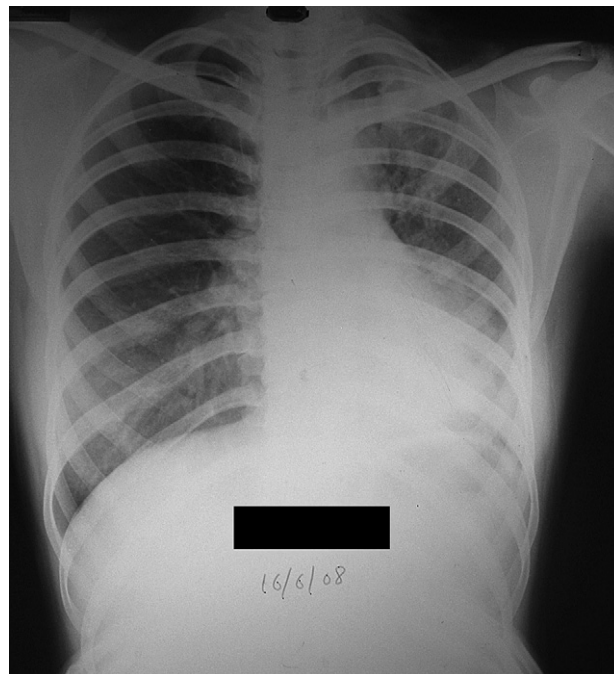


Fig. 3. Left lung partial expansion after the removal of clot.

examination of lungs from fatal hemorrhage cases.¹⁰ Most bleeds in pulmonary tuberculosis are caused by vascular erosion, where bronchial lesions will perforate or erode the adjoining vessel.¹⁰ In the above case, as there was no obvious cavity formation in the chest radiograph, probable etiology of the hemoptysis was the erosion of vessel adjoining to the pulmonary lesion in the left upper lobe.

Endobronchial obstruction due to blood clot is especially worthy of consideration in a patient following an episode of massive hemoptysis,¹ which had happened in this case as well. The first confirmed case of endobronchial obstruction from blood clot was described by Wilson in 1929. The patient was a 23-year-old woman

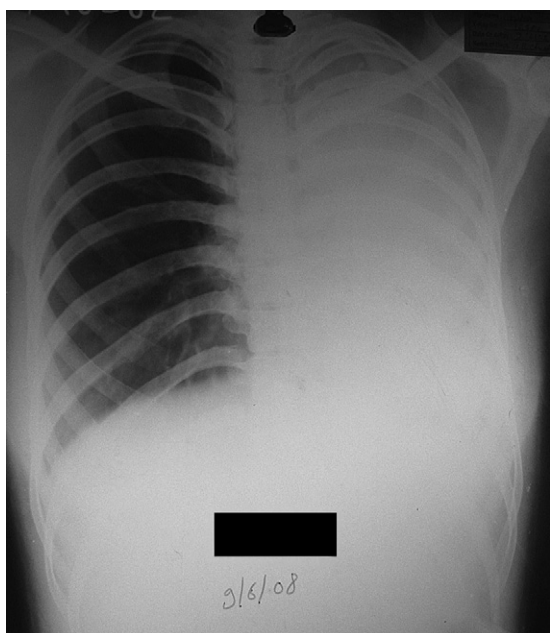


Fig. 2. Left lung complete collapse secondary to airway obstruction by a blood clot.

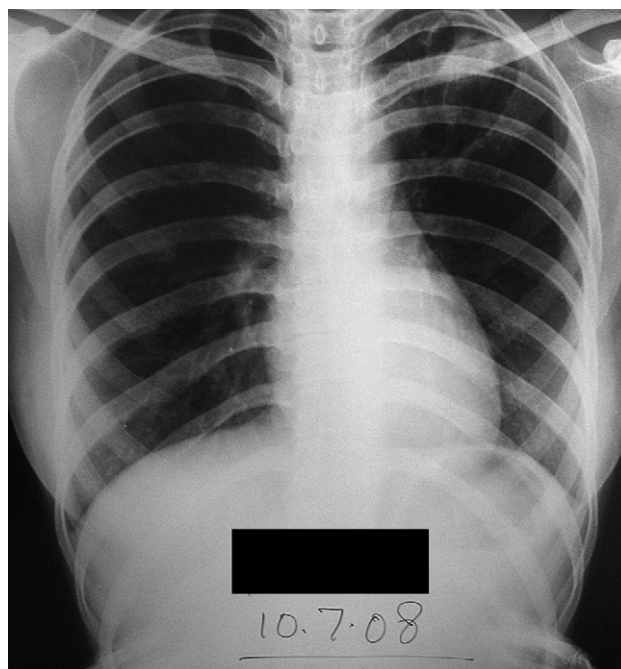


Fig. 4. Left lung complete expansion.

with tuberculosis and atelectasis. She had respiratory distress after 3 days of recurrent hemoptysis, and her chest radiograph revealed right middle and lower lobe collapse. Six days later, she expectorated a large bronchial cast composed of blood clot and her respiratory symptoms rapidly improved.¹³

Damage to a blood vessel results in vascular spasm to reduce blood loss while other mechanisms also takes effect; Blood platelets congregates at the site of damage and amass to form a platelet plug. This is the beginning of the process of the blood “breaking down” from its usual liquid form in such a way that its constituents play their own parts in processes to minimise blood loss. Blood clotting (technically “blood coagulation”) is the process by which (liquid) blood is transformed into a solid state. This blood clotting is a complex process involving many clotting factors (including calcium ions, enzymes, platelets, damaged tissues) activating each other. The three stages process includes formation of prothrombinase, which converts prothrombin (that is formed in the liver) into the enzyme thrombin. Thrombin converts fibrinogen (which is also a plasma protein synthesized in the liver) into fibrin. Fibrin is insoluble and forms the threads that bind the clot.

In a normal pregnancy, there is a marked increase in the pro-coagulant activity in maternal blood, characterized by elevation of factors VII, X, VIII, fibrinogen and von Willebrand factor, which is maximal around term. This is associated with an increase in prothrombin fragments (PF1 + 2) and thrombin–antithrombin complexes. There is a decrease in physiological anticoagulants manifested by a significant reduction in protein S activity and by acquired activated protein C (APC) resistance. The overall fibrinolytic activity is impaired during pregnancy, but returns rapidly to normal following delivery, thus decreasing bleeding complications in connection with delivery.^{2,3,5} Using 30 years of data, Heit and colleagues found that the risk for a first episode of venous thromboembolism is 5 times higher in the postpartum period than during the pregnancy. The risk for pulmonary embolism is 15 times greater during the postpartum period than during the pregnancy, which implies that women at high risk for venous thromboembolism may require special consideration for anticoagulation in the postpartum period.⁴

Hypercoagulable state is also seen in PTB patients with active PTB showing anaemia, leucocytosis, thrombocytosis, elevation in plasma fibrinogen, factor VIII, plasminogen activator inhibitor 1 (PAI-1) with depressed antithrombin III (AT III) and protein C (PC) levels and improve with treatment.¹³

Ethamsylate injection was also administered in the above case. Ethamsylate drug works, not by interfering with coagulation directly, but rather through increasing capillary vascular resistance and platelets adhesiveness. The drug is used for preventing and treating capillary haemorrhages associated with menorrhagia, haemoptysis, haematuria etc. Whether the blood clot formed in the above case, was the combined result of all these risk factors or it was due to slow accumulation of blood at the site of pulmonary lesion, with application of natural mechanism of clot formation, is a matter of research. Probably, it was the natural mechanism of the clot formation with the added effect of drug ethamsylate, which had resulted in the blood clot formation in this case, as the patient's coagulation profile was normal.

The initial effort at removal of the endobronchial clot should involve the flexible bronchoscopic evaluation with saline lavage and suctioning. If unsuccessful, the usual next step is forceps extraction through the working channel, either en bloc or in piecemeal fashion. The rigid bronchoscopy allows the clinician greater access for suctioning and forceps extraction. If brisk endobronchial bleeding occurs following clot removal, the rigid

bronchoscope offers superior airway management. Recently, topical thrombolysis has been used with success. Streptokinase has been used most often, in doses of 30,000–120,000 U; 30,000–60,000 U of streptokinase are mixed in 30–60 mL of normal saline, respectively, for a concentration of 1000 U/mL. Aliquots of 10–15 mL are applied, allowing 5–10 min between dosing to take effect.¹¹ Urokinase, the premixed formulation for central catheter clearance, has also been used with success in aliquots of 2500 U in 5 mL of diluent. Thomson has recommended the use of a plastic catheter, to more easily and accurately direct the lytic agent, onto the surface of the clot. Although recurrence of bleeding has not been reported with thrombolytics, it should be regarded as a potential consequence, and anticipated management needs should be considered in advance. In the above case, rigid bronchoscopy was successful in removing the blood clot, following failure with the flexible bronchoscope and no further obstructive event occurred.

Teaching points

1. Blood clot may occur following an episode of massive hemoptysis and may result in partial or complete atelectasis of lung.
2. Both pregnancy and tuberculosis are the hypercoagulable states and may result in deep vein thrombosis, warranting anticoagulation treatment in selective cases.
3. Ethamsylate should be used judiciously in the management of hemoptysis.
4. Blood clot can be removed by various interventional modalities, having their own merits and demerits.

Conflict of interest statement

None of the authors has a conflict of interest to declare in relation to this work under this sub-heading.

References

1. Arney KL, Judson MA, Sahn SA. Airway obstruction arising from blood clot: three reports and a review of the literature. *Chest* 1999;**115**:293–300.
2. Brenner B. Hemostatic changes in pregnancy. *Thromb Res* 2004;**114**(5–6):409–14.
3. Cerneca F, Ricci G, Simeone R, Malisano M, Alberico S, Guaschino S. Coagulation and fibrinolysis changes in normal pregnancy. Increased levels of procoagulants and reduced levels of inhibitors during pregnancy induce a hypercoagulable state, combined with a reactive fibrinolysis. *Eur J Obstet Gynecol Reprod Biol* 1997 May;**73**(1):31–6.
4. Heit JA, Kobbervig CE, James AH, Petterson TM, Bailey KR, Melton LJ. Trends in the incidence of venous thromboembolism during pregnancy or postpartum: a 30-year population-based study. *Ann Intern Med* 2005;**143**:697–706.
5. Hellgren M. Hemostasis during normal pregnancy and puerperium. *Semin Thromb Hemost* 2003 Apr;**29**(2):125–30.
6. Iseman M. Hemoptysis: origins and management. In: Wilkins LW, editor. *A clinical guide to tuberculosis*; 2000. p. 140–4.
7. Ran W, Garay S. Hemoptysis. In: BaC Little, editor. *A clinical guide to tuberculosis*; 1996. p. 392–3.
8. Rasmussen V. In: *Hemoptysis, namentlich der lethalen in anatomischer und klinischer Beziehung*. Hospital Tidende; 1868.
9. Sanyika C, Corr P, Royston D, Blyth DF. Pulmonary angiography and embolization for severe hemoptysis due to cavitory pulmonary tuberculosis. *Cardiovasc Intervent Radiol* 1999;**22**:457–60.
10. Thompson JR. Mechanisms of fatal pulmonary hemorrhage in tuberculosis. *Dis. Chest* 1954;**25**:193–205.
11. Thomson DB. Endobronchial streptokinase to dissolve a right mainstem clot. *Chest* 1986;**89**:904.
12. Turken O, Kunter E, Sezer M, Solmazgul E, Cerrahoglu K, Bozkanat E, et al. Hemostatic changes in active pulmonary tuberculosis. *Int J Tuberc Lung Dis* 2002 Oct;**6**(10):927–32.
13. Wilson JL. Hemoptysis is tuberculosis followed by massive pulmonary atelectasis. *Am Rev Tuberc* 1929;**19**:310–3.